exposed to 0.9 T showed a 7% increase in volume. Shortening was reduced by approximately 30% after recovery in both 0.6 T and 0.9 T groups. Thus, despite a 23% difference in size between groups, there was no difference in function.[3] This argues against a direct relationship between edema and dysfunction.

In Dr Lawton's study, function was measured only in the recovery phase, when cardiomyocytes had returned to their normal size and not during the time in which cells were swollen. Yet, in the clinical context, edema and dysfunction occur concurrently, and this has largely been the basis for a causal association to be drawn. Data we present from Langendorff-based analyses show only a limited association exists. An increase in water content of 3.3% was associated with a 9% reduction in developed pressure, which in this model is a modest decline. Developed pressure was stable for up to 20 minutes of hypotonic exposure and returned to baseline with isotonic reperfusion.[1] Our surgical modeling suggests that swelling is tolerated for several hours without functional effect. Thus, it is unlikely that the duration of hypotonic exposure, a further methodologic difference between our studies, was responsible for the lack of edema-related dysfunction.

It must be acknowledged that experimental separation of tonicity from ischemia, removing osmotic influences of the interstitial and vascular compartments, is highly artificial. Future use of human myocytes in this model will also be subject to the same caveats. It is likely that the dogma relating edema to dysfunction confuses *extracellular* water accumulation, as a result of capillary leak and renal effects of lower cardiac output, with *intracellular* water accumulation, which is well tolerated by the heart and quickly resolves through transmembrane water exchange.[4,5]

The most credible interpretation of data from both our groups is that the swollen heart of ''myocardial edema'' as observed by surgeons after long neonatal cardiac surgery actually represents the effects of myocardial dysfunction with the ventricles working at a higher intracavitary volume and reduced fractional shortening, almost all of which can be attributed to the effects of ischemia and reperfusion. The water content of the myocardium itself may be increased by a few percentage points, and this contributes to a decrease in cardiac performance by a similar amount.

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WHAT IS THE REAL RISK OF STENT-GRAFT INFECTION IN THE TREATMENT OF AORTOBRONCHIAL FISTULAS?

To the Editor:

A recent article by Hacker and colleagues[1] describes a single case report of aortobronchial fistula (ABF) in a patient who was successfully treated (27 years ago) for isthmic posttraumatic pseudoaneurysm by placement of thoracic endoprosthesis. The authors conclude by saying that ''endovascular intervention and stent grafting are feasible and should be the first option in the treatment of ABFs.''

We agree that ABF is a rare and fatal disease and most frequently associated with previous cardiac, vascular, or thoracic surgery. Surgical treatment is still characterized by high rates of mortality and morbidity. Our article[2] showed that it is not possible to identify a half-accurate diagnostic instrument: angiography, bronchoscopy, and transesophageal echocardiography are associated with a high rate of complications, including fatal hemorrhage. Findings suggestive of ABF on computed tomography scan include pseudoaneurysm, aortic anatomy abnormalities, lung parenchyma consolidation, and compression of bronchial tree.

Conventional open surgical correction involves a thoracotomy and has fairly high morbidity and mortality because of difficult operative dissection associated with reoperative surgery; in addition, patients may have severe comorbidities and present in rather poor health. Endovascular stent grafting provides a safe and reliable method to treat ABF.

One potential life-threatening complication is stent-graft infection; however, in our experience, no signs of infections were detected by postoperative scintigraphy with labeled leukocytes.

It is unclear why the infection rate for endovascular repair is minimal, but the stent graft remains in the center of the aneurysm sac well away from the actual fistula and source of contamination. Perhaps because there is minimal tissue trauma associated with the deployment of the stent graft, as opposed to open surgical repair, the excluded aneurysmal or pseudo-aneurysmal cavity is less likely to become contaminated or infected.

Postoperative antibiotic treatment differs completely among the various authors, both with regard to the antibiotic (cephalosporin, vancomycin,

antifungal) and the duration of therapy (lifelong antibiotic therapy?).

In our opinion, the open questions remain: (1) Which antibiotic therapy should be performed in patients with ABF treated with thoracic endovascular aneurysm repair? (2) How long should antibiotic therapy be continued?

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Reply to the Editor:

Bozzani and colleagues[1] state that surgical correction of an aortobronchial fistula, particularly open correction of a thoracic aneurysm, carries a fairly high postoperative incidence of stent-graft infection. To the contrary, minimal infection rates were observed after endovascular stent placement. The authors question whether antibiotic therapy should be administrated after this minimally invasive operational procedure.[1,2] There is scarce literature on immunologic consequences after stent implantation in humans. According to immunologic data obtained from patients undergoing heart operations with cardiopulmonary support and abdominal surgery, any operation performed in humans induces a state of immune suppression in vivo. Therefore, patients undergoing heart surgery (cardiopulmonary support) should receive aggressive 5-day antibiotic treatment in accordance with the insight of an induced ''systemic immune suppression'' after heart surgery.[3,4] In regard to the ongoing discussion of antibiotic treatment after endovascular stent implantation, the following approach seems to be feasible. Studies have to be initiated to investigate the immunologic consequence of open and endovascular stent implantation in humans (eg, abdominal aorta aneurysm repair, open, closed), and ''yes,'' antibiotic treatment should be applied for 4 to 5 days after endovascular stent placement to potentially ''prohibit'' pain from infection (local, systemic) in patients.

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A COMPLEMENTARY TECHNIQUE TO CARBON DIOXIDE DE-AIRING IN OPEN CARDIAC OPERATIONS?

To the Editor:

In a recent study Al-Rashidi and associates[1] concluded that ''bilateral … pulmonary collapse and successive filling of the lungs with … concomitant increase in mechanical ventilation during de-airing of the left side of the heart significantly reduces the number of systemic MES [microembolic signals]… and … air emboli.''

Inasmuch as the study's limitations were not mentioned, we would like to discuss a few:

1. Inasmuch as the patients were alternately allotted to control and study groups, the principle of randomization was ignored. Moreover, the principle of unbiased assessment was also compromised inasmuch as the single surgeon, who actively followed the degree of de-airing via transesophageal echocardiography (TEE) during the surgical de-airing maneuvers before the end of cardiopulmonary bypass (CPB), thus participated in the evaluation of the technique, which he himself had proposed.
2. The authors enumerate 9 exclusion criteria, including accidental opening of the pleural cavity and chronic obstructive lung disease, although all operations were performed by one highly experienced surgeon. Consequently, the generalizability of the results cannot but be very limited.
3. According to the Methods section, TEE and transcranial Doppler (TCD) measurements were only performed *after* CPB. However, in the Results section, the authors make comparisons with the number of MES *before* the end of CBP. Moreover, ''The aortic root was de-aired'' before release of the crossclamp only in the study group.
4. The possible risks of a left ventricular vent inserted through the apex of the heart (eg, bleeding and arrhythmias), a prerequisite of the technique, have not been mentioned.
5. In contrast to common practice as well as the study group, the control group patients had their lungs ventilated continuously during